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February 5, 2019

Amy L Fair RN
Simmons Hanly Conroy
One Court Street
Alton, IL 62002

Re: Grace Holland Webster
DOB: October 16,1929
DOD: July 8, 2016

In the United State district Court for the Eastern District of North Carolina
Civil Action No.: 2:17-CV-63-D.
Karen Cahoon, as executrix of the Estate of Grace Webster, Deceased, Plaintiff
v. Edward Orton, Jr. Ceramic Foundation, et al

Dear Ms. Fair,

In response to your request concerning the matter above-captioned I prepared this report according to the instructions received from your office regarding comments made by the court and related requirements of the applicable federal rules.

You have asked for statements as to: a) my compensation for testimony in this case which will be $600 per hour for trial and deposition testimony and $600 per hour for research and reports; b) a listing of all the cases in which I have testified, either at trial or by deposition in the last 4 years, and I have attached the most updated version of a report maintained in connection with this information; and c) my qualifications, including publications authored within the past 10 years, I have attached a copy of my current curriculum vitae, which contains that information, and I will further address my qualifications below.

I am a licensed physician and currently a Clinical Professor in the Division of Occupational and Environmental Medicine in the Department of Internal Medicine at the University of California San Francisco School of Medicine. Additionally, I have a Master of Public Health degree from the UCLA School of Public Health in Epidemiology and a private practice in Occupational Medicine and Industrial Toxicology. I am Board-certified by the American Board of Preventive Medicine in General Preventive Medicine and in Occupational Medicine.

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My private practice focuses on occupational medicine and industrial toxicology, providing consultative services to industrial and other clients regarding occupational medicine and industrial toxicology. This includes interpretation and application of OSHA Occupational Health Standards; workplace assessment and recommendations regarding existing health hazards; interpretation of industrial hygiene data with assessment of health risk; interpretation of biological monitoring and medical surveillance data with recommendations, where appropriate, for further actions regarding health hazard identification and control; analysis of injury and exposure data; development of programs for management and employee training in medical, safety and industrial hygiene; and research and investigation in industrial toxicology including investigation of illness clusters and scientific literature review.

I have researched and published numerous articles on occupational health and industrial toxicology. This research includes the area of asbestos, what was known about the health hazards of asbestos, and when such information would have been reasonably known.

I have been qualified as an expert and have testified in numerous asbestos cases regarding the above, especially regarding the issue of when knowledge of the health hazards of asbestos became available. This subject is known as "asbestos historical state of the art". I have reviewed historical scientific and medical literature regarding asbestos disease hazards at the request of defense and plaintiffs' attorneys beginning in the early 1980s for providing asbestos state of the art testimony at trial in asbestos personal injury and wrongful death cases and other asbestos related litigation. I am qualified and routinely testify as to the medical issues involved in these cases, including but not limited to medical causation, asbestos risks and disease, epidemiology, state of the medical and scientific art concerning asbestos-related diseases at relevant times, the nature and use of asbestos products, and the individual's exposure to asbestos. I am qualified and often testify as to asbestos products defects, the release of asbestos fibers from asbestos products, disease causing potential of various asbestos products, asbestos health hazards, industry awareness, and state of the art relating to the hazards of asbestos, applicable rules and regulations, and other industrial hygiene related issues.

You have asked for a statement of the opinions I will express in this case and the basis for them. In that regard, I am of the opinion as to the medical issues involved in this case, that medical causation was established, in whole, in part or in combination with, the inhalation of asbestos fibers. In my view, the knowledge of and risk(s) for the pertinent asbestos-related diseases was demonstrated by the related epidemiologic and exposure related publications concerning such asbestos-related diseases. This information was available to the scientific and medical community, thus also to the public. This information would include the state of the medical and scientific art concerning asbestos-related diseases at relevant times and information regarding the nature and use of asbestos or asbestos-containing materials or products.

My opinions can also be expressed about specific products and/or applications. Such products and/or applications in my opinion presented and/or still present a risk of asbestos exposure to those using such products and/or those being around others who were using or manipulating such products. In my opinion this is specifically true as to the products or applications attributable to each of the defendants in this case whose products or activities are alleged to have caused exposures in this case.

I am of the opinion that there are defects in asbestos or asbestos-containing products, such as those mentioned above, which include, but are not limited to, the presence of asbestos itself or the release of asbestos fibers from such asbestos products. I believe and will testify that these defects relate to the disease potential of the various asbestos products, and their ability to cause disease or create asbestos health hazards. I will testify there existed an awareness of such diseases and/or hazards within the asbestos and related industries generally, or defendants specifically, and that this awareness was either known or knowable to such parties. This was because the information concerning asbestos hazards was available to and shared by members of the relevant industries [e.g. asbestos mining and manufacturing companies; asbestos product users such as railroads, petroleum, equipment manufacturers] as well as in the public domain. The latter is evidenced by documents which describe and memorialize the state of the art relating to the hazards of asbestos. These documents include applicable governmental rules and regulations, and industrial hygiene, scientific, medical and related publications and articles discussing and addressing such issues.

As to the data or other information, in addition to case-specific material, which I have considered in formulating my opinions in this case, this would be, generally, the body of medical, scientific and epidemiological studies, case reports, investigations, government documents, public media and industry documents published and reported upon since the late 1890's in the USA, Britain, Germany, Italy, and other industrial areas abound the world. Attached is a copy of the bibliography I compiled that includes the documents that form the basis of the opinions I will express at trial.

Based on my above-mentioned experience and training, and historical review, in as further support for my opinions in this case to those stated above, I would observe that it is my opinion that the medical and scientific literature makes it clear that, at least as early as 1931, it was known in the medical and scientific community that breathing asbestos dust was harmful and dangerous to human health. As stated by Dr. Frederick Willson in 1931, "We do know, however, that breathing of dust under the following conditions is seriously harmful: . . . asbestos and every operation in which it is used." (Willson, Frederick, The Very Least An Employer Should Know About Dust And Fume Diseases, Safety Engineering, November 1931, Volume 62(5), pp. 317-318, emphasis added.) (This article is fully incorporated herein by this reference.) Additionally, the fact that asbestos exposure causes asbestosis, and the need for safety precautions, including masks, respirators, education, ventilation, dust control, and substitution, to prevent asbestos-related diseases, was known as early as the 1930s, as referenced in articles contained in my Bibliography, including Merewether ERA, Price CW, Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry, His Majesty's Stationery Office, London, 1930, pp. 1-34. Many of the preventive techniques to address asbestos exposures are still in use today as standard industrial hygiene. Further, based upon my research, education, experience, and the articles referenced in my bibliography, it is my professional opinion that it was clear by 1952 that, regardless of the setting, a person exposed to

airborne asbestos was at an increased risk of developing cancer.

Based on my above-mentioned experience and training and historical review, in my opinion a summary of only a few of the many articles I have studied indicates the following, which are also my opinions about, the historical state of knowledge about the hazards of asbestos:

a. As early as 1898, the Annual Report of the Chief Inspector of Factories and Workshops in England identified increased health problems among workers in asbestos textile mills.

b. In 1924, Cooke wrote an article in the British Medical Journal titled "Fibrosis of the Lungs Due to the Inhalation of Asbestos Dust".

c. In 1931 an article was published in the magazine Safety Engineering, which was a publication intended for people who had responsibility for preventing injury and illness. This article, titled "The Very Least an Employer Should Know About Dust and Fume Diseases," lists several conditions under which breathing of dust is seriously harmful. That list includes the entry: "Asbestos and every operation in which it is used."

d. In 1934, in an article titled "Pulmonary Asbestosis" published in The Lancet, two physicians reported 100 cases of people with asbestosis. The occupations of the people involved revealed that it was not only workers in the asbestos textile factories who developed asbestosis, but also people who worked with asbestos in other applications. The significance of the article is that it demonstrated that what was important was not the job or its location, or the product involved, but the fact that one had inhaled asbestos dust.

e. A publication by the Commonwealth of Pennsylvania, Department of Labor and Industry, in 1935, titled “Asbestosis," was remarkable because it contained a bibliography of over 100 reference works pertaining to asbestos related disease. The significance of the article is that as of 1935, a doctor in the U.S. in a relatively small town, Harrisburg, PA., could become aware of over 100 articles published about asbestos and disease and written in various languages.

f. In 1944 an editorial in the Journal of the American Medical Association identified asbestos as one of the causes of environmental cancer. The significance of the article is that by this time in 1944, credible and respected authorities in the medical community considered that asbestos was suspected of or known to cause cancer.

g. In 1949 an article appeared in Scientific American titled "Cancer and the Environment." It was one of the first articles to appear in the popular media to discuss the subject of asbestos being a possible cause of cancer. I have also found reference to asbestos as a cause of cancer in newspaper articles as early as 1949.

h. In 1950, Dr. Hueper, the Chief of the Carcinogen Study Section, National Cancer Institute, National Institute of Health, published a monograph titled "A Methodology for Environmental and Occupational Cancer Surveys" in Public Health Technical Monograph No. 1, 1950. In that article he listed agents, chemicals, metals, dusts, etc. which were known to cause cancer. Among those substances was asbestos. Under asbestos he listed various asbestos-related trades or jobs that he considered to be at increased risk for cancer because they involved asbestos

exposure. The significance of the article is that it reveals that there was a cancer concern not only for the asbestos factory workers, but for other trades exposed to asbestos working with asbestos containing products. Among the at-risk trades identified by Dr. Hueper were: asbestos construction material workers, asbestos insulation workers, asbestos brake lining workers, people that use asbestos brake lining, carpenters, plumbers, roofers, gasket makers, insulation workers (pipe and boiler), and pump packing mechanics.

i. By 1952 the Encyclopedia Britannica contained an entry indicating that asbestos is a cause of cancer.

j. By 1958 the American Conference of Governmental Industrial Hygienists had established a maximum atmospheric concentration for asbestos dust of five million particles per cubic foot of air. This amount of asbestos in the air was then known not to be visible and could only be detected by air sampling measurements.

k. In 1960, Dr. Wagner published a study of mesothelioma victims in South Africa. His study unequivocally established asbestos as the cause of mesothelioma.

l. In 1964 Dr. Selikoff published his seminal article on asbestos disease in insulation workers. He performed the first large scale asbestos mortality study and reported increased rates of death from lung cancer, mesothelioma, asbestosis, and gastrointestinal cancer. In addition to its scientific contribution, it was politically and socially significant because it received widespread media exposure. It was reported to American newspapers by the Associated Press wire service and brought public attention to the health risks associated with asbestos.

Based on my above-mentioned experience and training and historical review, in my opinion I can further state the following facts about the historical knowledge of the dangers of asbestos:

A) By the 1960s, there were at least 300 articles published in English concerning the hazards of asbestos. There were a similar number published in foreign languages.

B) In the late 1920s and certainly by the 1930s it was clear that breathing asbestos dust caused asbestosis. Dr. Merewether's report in 1930, published by the British government, lists various actions that he recommended to prevent the disease. Other publications in the 1930s indicate clearly that people at that time understood that they could prevent the disease by preventing individuals from breathing the dust. They suggested this could be done by either eliminating the dust at its source or by providing gas masks as breathing protection. In the late 1940s and early 1950s it became clear that asbestos could cause asbestosis in a large variety of settings. It was demonstrated clearly in the literature by the early 1950s that asbestos related disease could occur in any locale or with any task or with any product involving sufficient asbestos exposure.

C) Information regarding asbestos hazards, as well as remedial steps to eliminate or reduce those hazards, was not confined to those involved in academia or medical research. The availability of information to business and industry is illustrated by documents generated by private industry dating from the 1930s:

a. The 1935 Minutes of the Medical and Surgical Section of the American

Association of Railroads show that the physicians who were responsible for medical issues among railroad employees discussed exposure to asbestos and related risk to railroad workers. They included discussion of preventive measures that are still used today, including: using masks to filter the air; using techniques to wet down the dust lying on the floor, so it doesn't get re-circulated; using apparatus that would pull the dust away from the worker, and doing medical exams on the workers to determine if they are sufficiently protected from asbestos so as to not develop asbestos related disease. The historical state of the art evolved in such a way that it was clear in the early 1950s, that people performing tasks or in situations that generated airborne asbestos, regardless of the product involved, were going to be at risk for asbestos-related disease.

b. In 1937, Roy Bonsib conducted a study of dust hazards present in the oil refinery setting on behalf of Standard Oil Company of New Jersey. He included asbestos as a hazardous dust and noted the greater level of hazard presented by the removal of asbestos insulation materials. He recommended the use of well-recognized procedures to minimize the creation of dust and reduce or eliminate exposures to hazardous dust (asbestos) including engineering controls, wet down, containment, isolation of work, and the use of respirators.

c. In addition to what was knowable through medical and industrial hygiene literature and industry publications, businesses in California for example were additionally on notice as to the hazards of asbestos from an early point in time by various governmental regulations and orders. Commencing in the 1930s, the California General Industry Safety Orders prescribed procedures for minimizing exposure to asbestos dust including requirements for exhausting asbestos dust, dust suppression procedures, and isolation of dust-creating work from other workers. After the creation of OSHA in 1971, federal exposure standards regarding asbestos were imposed commencing in 1971. These regulations were published expressly to notify those working with asbestos containing material of its hazards and the precautions which were necessary to be employed to reduce the risk of harmful exposures. The regulations included descriptions of the previously-described protective measures that should be followed to eliminate or minimize exposures, and which had been recognized and recommended since at least the 1930s.

Case Specific Opinions

At your request, I have reviewed and considered case specific documents forwarded by your office for rendering an opinion regarding the possible presence and/or cause of any asbestos-related disease suffered by Grace Webster. In addition, I have drawn upon my general knowledge of the medical and scientific literature pertaining to asbestos related disease as well as my experience as a practicing specialist in occupational medicine and industrial toxicology for over 40 years. The case specific documents I reviewed include the following:

1. Medical records from treating physicians.
2. Work history prepared by plaintiff’s attorney based on deposition and other evidence.
3. Death certificate.
4. Deposition testimony of Karen Cahoon dated November 27, 2018, including attached exhibits 2 (pictures related to decedent’s work and potential exposures) and 3 (Analysis of Vermiculite Packing Material of Orton Pyrometric Cones, July 17, 2018).
5. Social Security records of Grace Webster.

Grace Webster was 86 years old when she was evaluated by her physician in June 2015 due to progressive dyspnea and right upper quadrant abdominal pain. She was found to be hypoxic and treated with pulmonary rehabilitation without improvement. As of November 2015 she had developed dyspnea with minimal exertion. She was evaluated by her physician on December 2, 2015 due to complaints of persisting and worsening abdominal pain over several months. She also reported exertional dyspnea, fatigue and fever. Abdominal CT scan was performed December 3, 2015 due to complaints of abdominal pain. A large right pleural effusion and a right medial soft tissue thoracic mass were observed.

Right thoracentesis was performed on January 8, 2016 and January 29, 2016; both were negative for malignancy. Chest CT scan performed on January 16, 2016 again showed a large right pleural effusion and nodular pleural thickening suspicious for pleural malignancy. Chest x-ray on February 22, 2016 showed a large right pleural effusion and totally opacified right hemithorax.

Right video assisted thoracoscopy, pleural biopsy, total pleural decortication, and pleural fluid drainage were performed on March 14, 2016. The pathology diagnoses of the pleural fluid and tissues was “malignant mesothelioma, epithelioid type”. Mesothelial tumor involved both the visceral and parietal pleura. Bedside talc pleurodesis was performed on March 18, 2016. As of March 20, 2016 her physician’s prognosis was “poor” due to the diagnosis of the mesothelioma.

Chest x-ray on May 24, 2016 showed evidence of a right pulmonary mass and persisting right pleural effusion. As of June 14, 2016 she was being treated (for mesothelioma) with analgesics, oxygen and otherwise palliative care. She died on July 8, 2016.

Grace Webster’s past medical history includes hypertension, gout, allergic rhinitis, anxiety, hypothyroidism, chronic kidney disease, and gastroesophageal reflux disease. Past surgeries include cholecystectomy, retinal detachment repair, appendectomy, breast biopsy and hysterectomy. She was a former smoker and quit in approximately 2011. Smoking does not cause or contribute to the cause of mesothelioma. Medications in use prior to mesothelioma diagnosis included paroxetine, clonazepam, atenolol, amlodipine, Synthroid, Uloric, omeprazole, Patanase, Atrovent, Systane ophthalmic solution, aspirin, Atrovent, ibuprofen, Patanol ophthalmic, and vitamins.

Grace Webster’s family medical history includes diverticulitis, hypertension, and stroke.

Grace Webster had regular, repeated, intense and proximal exposure to asbestos over several decades beginning in 1968 and ending no later than the early 2000’s. Her exposure resulted from her unpacking and handling the asbestos contaminated vermiculite packing material in which Orton Pyrometric Cones were sold to her. The small cones were used by her in the kilns used to bake ceramics that she produced in her work as a teacher of ceramics in her home-based business.

The pyrometric cones were sold to her in small boxes that were filled with the asbestos contaminated amphibole vermiculite packing material in which the cones were randomly dispersed. She disturbed the vermiculite repeatedly in pulling the cones out of the boxes and sometimes dumping the box’s contents of vermiculite and cones in order to find the cones more easily. Disturbing the vermiculite created visible airborne dust that contaminated her work area and which she brushed off with her hands and swept up with a broom. She performed these tasks

on a daily basis and used approximately 1 to 2 boxes (50- 100 cones) monthly. This work was performed in small rooms without exhaust ventilation.

It has been well established in the medical, scientific, and industrial literature since the 1930s that inhaling dust containing asbestos fibers could lead to disabling and/or fatal asbestosis. Cases of lung cancer associated with the inhalation of asbestos were also first reported in the 1930s, and, by the early 1950s, it was well understood that the inhalation of asbestos fibers caused lung cancer. Inhalation of asbestos dust as a cause of mesothelioma has been accepted by the medical and scientific community since 1960.

Asbestos causes a variety of injuries to the human respiratory system, including non-malignant diseases and several cancers. Asbestos is the only known cause of most, if not all, U.S. cases of mesothelioma, a fatal cancer that arises in the pleura, peritoneum, pericardium and other mesothelial sites. Asbestos also causes cancers in various parts of the body, including the lungs, larynx, ovaries, colon, and stomach.

I do not have, and never have agreed with, any so-called "every fiber" "every exposure", "each and every exposure", "any exposure", or "cumulative exposure" "theory". My medical causation opinions regarding how an identified dose contributes to cause an asbestos-related disease in a particular individual who has been diagnosed with such disease are complex and not reasonably described with short catch-phrases. My medical causation opinions are set forth as a whole in the following paragraphs:

In 1997, nineteen "pathologists, radiologists, occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists and clinical and laboratory scientists" from eight countries, met in Helsinki, Finland, to discuss and publish consensus attribution criteria for the specific causation of the primary asbestos diseases. The resulting consensus document, and its 2014 update, represent the opinions of these international asbestos disease experts on specific asbestos causation and has been referred to by physicians for specific attribution of diseases including asbestosis, several asbestos induced cancers, and mesothelioma.

The Helsinki Consensus Report recognized that very low doses of asbestos can cause mesothelioma. It also recognizes that all asbestos fiber types can cause mesothelioma and other cancers. Markers of asbestos exposure are not essential to attribute a mesothelioma or other cancers to asbestos exposure. In the absence of such markers, "a history of significant occupational, domestic, or environmental exposure to asbestos will suffice for attribution." The group further concluded that, "In some circumstances, exposures such as those occurring among household members may approach occupational levels." Asbestos, asbestosis and cancer: The Helsinki criteria for diagnosis and attribution. (Scand J Work Environ Health 1997:23(4):311-316.)

Mesothelioma, lung cancer and other cancers caused by asbestos exposure are cumulative dose-response diseases of long latency. Mesothelioma is a signature disease for asbestos exposure; identified exposure to asbestos is the only generally accepted cause for mesothelioma occurring in North America. Numerous regulatory and scientific agencies that have considered this issue have indicated that low dose exposures, such as household and neighborhood, cause mesothelioma and that a safe level of asbestos exposure related to any cancer has not been established. These include, but are not limited to, the World Health Organization, the World

Trade Organization, the International Agency for Research on Cancer, the Occupational Safety and Health Administration, the National Cancer Institute, and the Environmental Protection Agency. The scientific research on the relationship between asbestos exposure and mesothelioma is well established, and it consistently shows that mesothelioma can be caused by low lifetime doses and infrequent exposures. The first major epidemiologic study of mesothelioma published in the peer reviewed literature, the 1960 Wagner study, contained both occupational and environment cases with the asbestos exposures ranging widely in both proximity and intensity. (Wagner JC, Sleggs CA, Marchand P (October 1960). Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. Br J Ind Med. 17: 260-71.) Similarly, "safe" exposure limits have not been established for the other asbestos related cancers.

After a patient is diagnosed with mesothelioma, one of the first questions to resolve is where and when asbestos exposure occurred. Because asbestos dust is so strongly associated with mesothelioma, proof of an identified exposure to asbestos dust within the latency period is proof of specific causation in a given case. It is generally accepted in mainstream medicine that mesothelioma is a very low dose disease, with no known minimum threshold of lifetime exposure to asbestos below which there is no risk. Even an exposure below .2 fiber- cc/ years can be a significant contributor to the development of the disease. A recent example of this is provided in Offermans et al 2014 study of 58,279 men that found a statistically significant risk of mesothelioma in relation to asbestos exposure at a median lifetime cumulative exposure of 0.2 f-cc/years. Offermans NSM, et al, "Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherlands Cohort Study." JOEM 2014; 56:6-19.

The scientific and medical community has yet to determine a level of lifetime exposure to asbestos below which mesothelioma and other cancers will not occur. There have been numerous cases in the literature of low and/or distant exposures causing mesothelioma. These cases include, but are not limited to: short, high intensity exposures; secondary exposures such as household members exposed to asbestos contaminated work clothing brought home by another household member; and low-level environmental exposures wherein the victims lived considerable distances (up to ½ mile) from the source of exposure. It has been repeatedly and consistently demonstrated in the medical and scientific literature that family members exposed to relatively low-levels of asbestos dust from laundering a worker's clothing have a significant and increased risk of developing mesothelioma.

In addition to the more recent Offermans study described above, there are other peer review published studies that demonstrate an excess risk for mesothelioma at exposure levels at or below .1 fiber cc/ years. A large case control study by Iwatsubo et al., found excess of pleural mesothelioma in the lowest exposure group, with an estimated total/lifetime exposure of 0001-.49 fiber-cc/years. Iwatsubo, et al., "Pleural Mesothelioma: Dose Response Relation at Low Levels of Asbestos Exposure in a French Population-based Case Control Study," Am. J. of Epi. 148: 133-142 (1998). Rodelsperger also concluded that there was a distinct dose-response relationship even at low levels of asbestos exposure, with exposures from 0 to .15 fiber-cc/ years showing a significantly increased risk of mesothelioma. Rodelsperger, et al., "Asbestos and Man-Made Vitreous Fibers as Risk Factors for Diffuse Malignant Mesothelioma: Results from a German Hospital-Based Case-Control Study," Am. J. Industrial Medicine, 39:262-275, at 272 (2001).

Additionally, it is a misstatement to categorically assert that exposures to asbestos below 0.1 fiber cc/years do not contribute to the development of mesothelioma or other asbestos cancers. Even if there were a threshold of exposure below which mesothelioma or cancer did not occur, once that threshold is exceeded, each identified exposure to asbestos would increase the risk of development of mesothelioma, even if the identified exposure to a particular product or class of products was below 0.1 fiber-cc/years. For example, if over a person's lifetime, they encountered 0.05 fiber-cc/years exposure to asbestos associated with each different asbestos containing product to which they had exposure, and if they had contact with 1000 different brands of asbestos products, each for a different week, their cumulative asbestos exposure would equal 50 fiber-cc/years occurring over a 20-year period. There is no question that a 50 fiber-cc/year cumulative lifetime asbestos exposure places one at high risk of mesothelioma and other cancers.

The exposure analysis is patient specific, but in general, all identified exposures to asbestos in an asbestos cancer patient's history, regardless of fiber type, if prior to a sufficient minimum biologic latency of 10-15 years prior to diagnosis, contributed to the total asbestos fiber burden that, in turn, caused the mesothelioma or other asbestos cancer. An identified asbestos exposure from the patient's occupational and environmental would be: (1) a well-characterized source of asbestos; (2) an activity disrupting that source such that inhalable airborne asbestos fibers are generated; and (3) add to the body's burden of asbestos. As such, not all exposures automatically qualify as significant factors, because not every exposure generates significant concentrations of asbestos fibers. Nonetheless, there is no established lower limit or threshold of asbestos exposure responsible for causing mesothelioma or other asbestos cancers. No identified exposure to asbestos that meets the above criteria can be excluded from the causal dose of asbestos that contributed to cancer causation.

The low dose aspect of mesothelioma has been borne out in decades of human epidemiological studies, case reports, and laboratory research. There is no scientific requirement that the identified exposure last a certain amount of time, be of a given intensity, or occur with a certain regularity. In an individual who gets a clinical mesothelioma, after accounting for minimum biologic latency to occur, all prior identified exposures to asbestos experienced by that person with mesothelioma are a significant contributing factor in the development of the disease. The risk increases and accumulates with each identified exposure. There is no way to establish which identified exposure increment or constituent caused the changes seen. Therefore, in someone diagnosed with mesothelioma, after accounting for minimum biologic latency, all identified exposures to asbestos, regardless of fiber type or quantitative dose, play a role in the development of subsequent mesothelioma. The same analysis applies to asbestosis as well as other asbestos-caused cancers, although there may be additional contributors to causation such as alcohol or cigarette smoke.

Vermiculite from the Libby Montana mine contains tremolite, winchite and richterite noncommercial fibrous asbestos amphiboles. Studies of exposure to these vermiculite associated amphibole asbestos fibers have demonstrated their causation of malignant mesothelioma. Attached is a bibliography listing studies and documents upon which I rely for my opinions regarding exposure and disease associated with Libby vermiculite and its associated fibers. IARC 2012 and EPA 2014 are authoritative and detailed summaries of the available evidence regarding the association between fibrous asbestos amphibole contaminated Libby vermiculite exposure and asbestos related disease, including mesothelioma.

There is no way of predicting a level of asbestos exposure that is safe for any particular individual. Individual physiological responses vary. If a person is diagnosed with a disease known to be caused by asbestos and who had sufficient asbestos exposure to cause or contribute to the cause of the disease, asbestos causation is established. However, assuming latency requirements are met, there is no scientific basis upon which anyone can look back and exclude some identified exposures and include other identified exposures as being causally related to the asbestos-related disease which the individual has incurred. All identified exposures, regardless of fiber type, play a role in the development of the subsequent mesothelioma or other asbestos cancer. Although prospective risk of future mesothelioma and cancer development in an exposed "healthy" person is proportional to cumulative dose, in a person who has been diagnosed with an asbestos disease, it is the entire cumulative lifetime dose (after accounting for minimum latency) that has combined to cause the disease.

It is analogous to smoking cigarettes in that a physician will ask how many packs the individual smoked daily and over how many years to determine the number of "pack-years." Then that total (cumulative dose) will be correlated to the risk of a smoking related disease such as lung cancer or vascular disease. The physician is not interested in how many cigarettes the individual smoked of each brand of cigarettes during each year during his smoking history. The physician relates the total cumulative cigarette dose to the risk of causation of a smoking induced cancer or other disease. The same is true with asbestos - the total identified dose is considered. Science is unable to determine the points in time, circumstances, or events during the period of a cumulative exposure at which malignant cellular change begins. I am not aware of any scientific process or published peer-reviewed article that enables us to identify the precipitating events or points in time wherein the mesothelioma or carcinogenic change begins such that we could retrospectively identify a specific causative exposure by date, product, or activity and such that we could eliminate all other asbestos exposures from contributing to causation of the cancer or mesothelioma.

In conclusion, scientific authorities agree that asbestos in the form of chrysotile, amosite, crocidolite, anthophyllite and tremolite cause asbestosis, mesothelioma and other cancers. Furthermore, asbestos containing vermiculite has been well-characterized and accepted as a cause of malignant mesothelioma. The prospective risk of developing an asbestos disease is proportional to the amount of one's lifetime asbestos exposure, given latency requirements. Asbestos related diseases are cumulative identified dose response diseases. Asbestos-related diseases are not caused by one fiber or one isolated exposure. Most often, individuals who have developed asbestos-related diseases such as mesothelioma or lung cancer have encountered hundreds, thousands, or hundreds of thousands of discrete and identifiable exposures during their lifetime that combine to cause the asbestos-related disease. Whether the exposures are categorized by date of occurrence, product type, commercial entity, location, employer, activity, task, or otherwise, it is the totality of all such identified exposures that mainstream medicine and science correlates with prospective risk of disease as well as which when combined are deemed the cause of a diagnosed asbestos related disease.

It is not scientifically feasible to eliminate the causal contribution from any specific exposure or group of exposures over time; there is no scientific method to determine the individual impact on causation of each exposure event. The scientific and epidemiologic literature that has established asbestos as a cause of asbestosis, mesothelioma and other cancer is based in part on measures of lifetime asbestos doses.

Based on the information available to me, it is my opinion to a reasonable degree of medical certainty that Grace Webster had a pleural mesothelioma that proved fatal. Her mesothelioma was caused by her frequent, repeated, excessive and proximal asbestos exposures that resulted from her work with and around amphibole asbestos containing vermiculite packing material. Her asbestos exposure was the direct and only cause of her mesothelioma.

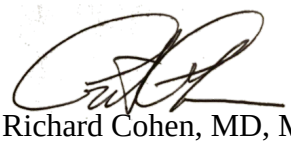
To a reasonable degree of medical certainty, exposure to asbestos was the proximate cause of the mesothelioma. Her mesothelioma was the result of no other causes revealed by her employment and medical history.

To a reasonable degree of medical certainty her mesothelioma was the predominant cause of her death.

The attached three bibliographies (Richard Cohen bibliography – 01 – 22 – 19; Mesothelioma; and Vermiculite include the non-case specific studies and documents upon which I rely for my opinions in this case.

Should you have any questions, please do not hesitate to contact me.

Sincerely,



Richard Cohen, MD, MPH

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382 - Musk, et al - Risk Factors for Malignant Mesothelioma in People with No Known Exposures to Asbestos - AJIM - 2017

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384 - News from the CDC - Asbestos Exposure Still Poses Occupational Health Risks - JAMA - 2017

385 - Olsson, et al - Exposure-Response Analyses of Asbestos and Lung Cancer Subtypes in a Pooled Analysis of Case-Control Studies - 2017

386 - Jiang, et al - Hand-Spinning Chrysotile Exposure and Risk of Malignant Mesothelioma, A Case-Control Study in Southeastern China - 2018

387 - Millette, et al - Microscopical Analyses of Asbestos-Cement Pipe and Board - 2018

388 - Micallef, et al - Occupational Exposures and Cancer, A Review of Agents and Relative Risk Estimates - 2018

389 - Xu, et al - Association between Mesothelioma and Non-Occupational Asbestos Exposure - 2018

Mesothelioma Index

Richard Cohen MD MPH
January 18, 2019

001 - Mallory, et al - Case Records of the Massachusetts General Hospital, Weekly Clinicopathological Exercises - 1947

002 - Sleggs, et al - Diffuse Pleural Mesotheliomas in South Africa - 1961

003 - Owen, W.G. - Diffuse Mesothelioma and Exposure to Asbestos Dust in the Merseyside Area - 1964

004 - Elmes & Wade - Relationship Between Exposure to Asbestos and Pleural Malignancy in Belfast - 1965

005 - Selikoff, et al - Relation Between Exposure to Asbestos and Mesothelioma - 1965

006 - Gilson, J.C. - Asbestos Cancer, Past and Future Hazards (Abridged) - 1973

007 - Webster, Ian - Asbestos and Malignancy - 1973

008 - Greenberg & Davies - Mesothelioma Register 1967-68 - 1974

009 - Zielhuis, et al - Pleura Mesothelioma and Exposure to Asbestos - 1975

010 - NIOSH - Studies of Human Population Carcinogenicity - 1 Day Exposure - 1976

011 - Graham, et al - Cancer in Asbestos-Mining and Other Areas of Quebec - 1977

012 - Sebastien, et al - Asbestos Retention in Human Respiratory Tissues, Comparative Measurements in Lung Parenchyma and in Parietal Pleura - 1980

013 -Tagnon, et al - Mesothelioma Associated with the Shipbuilding Industry in Coastal Virginia - 1980

014 - Vogelzang, et al - Malignant Mesothelioma: The University of Minnesota Experience - 1984

015 - Suzuki, et al - Translocation of Inhaled Asbestos Fibers From the Lung to Other Tissues - 1991

016 - Murai & Kitagawa - Asbestos Fiber Analysis in 27 Malignant Mesothelioma Cases - 1992

017 - Magnani, et al - A Cohort Study on Mortality Among Wives of Workers in the Asbestos

Mesothelioma Index

Richard Cohen MD MPH
January 18, 2019

Cement Industry in Casale Monferrato, Italy -1993

018 - Pott - Chrysotile Use and Carcinogenicity in Germany and a Comparison with Animal Studies - 1993

019 - Battifora & McCaughey -Tumors of the Serosal Membranes - 1994

020 -Dail & Hammar - Pulmonary Pathology book excerpts - 1994

021 - Kogan & Kikitina - Solubility of Chrysotile Asbestos and Basalt Fibers in Relation to Their Fibrogenic and Carcinogenic Action - 1994

022 - Meurman, et al - Incidence of Cancer Among Anthophyllite Asbestos Miners in Finland - 1994

023 - Spirtas, et al - Malignant Mesothelioma: Attributable Risk of Asbestos Exposure - 1994

024 - Smith & Wright - Chrysotile Asbestos is the Main Cause of Pleural Mesothelioma - 1996

025 - McDonald, et al - Mesothelioma in Quebec Chrysotile Miners and Millers, Epidemiology and Aetiology - 1997

026 - Iwatsubo, et al - Pleural Mesothelioma, Dose-Response Relation at Low Levels of Asbestos Exposure in a French Population-Based Case-Control Study - 1998

027 - Suzuki & Yuen - Asbestos Fibers Contributing to the Induction of Human Malignant Mesothelioma - 2002

028 - Pan, et al - Residential Proximity to Naturally Occurring Asbestos and Mesothelioma Risk in California - 2005

029 - Suzuki, et al - Short, Thin Asbestos Fibers Contribute to the Development of Human Malignant Mesothelioma, Pathological Evidence - 2005

030 - Dodson & Hammar - Asbestos: Risk Assessment, Epidemiology, and Health Effects - 2006

031 - Mark & Kradin - Patholgical Recognition of Diffuse Malignant Mesothelioma of the Pleura, The Significance of the Historical Perspective as Regards this Signal Tumor - 2006

032 - Sinninghe, et al - Environmental Exposure to Asbestos in the Area Around Goor has been

Mesothelioma Index

Richard Cohen MD MPH
January 18, 2019

Establshed as the Cause of Pleural Mesothelioma in Women (Abstract) - 2007

033 - Welch, L. - Asbestos Exposure Causes Mesothelioma, But Not This Asbestos Exposure - 2007

033.1 - Dodson, et al - A Technical Comparison of Evaluating Asbestos Concentration by PCM, SEM, and ATEM as Illustrated From Data Generated From a Case Report - 2008

034 - Goodman, et al - Ionizing Radiation: A Risk Factor for Mesothelioma - 2009

035 - Harding, et al - Mortality Among British Asbestos Workers Undergoing Regular Medical Examinations (1971-2005) - Occup. Environ. Med - 2009

036 - Loomis, et al - Lung Cancer Mortality and Fibre Exposures Among North Carolina Asbestos Textile Workers - Occup. Environ. Med - 2009

037 - Pira, et al - Mortality From Cancer and Other Causes in the Balangero Cohort of Chrysotile Asbestos Miners - Occup. Environ. Med - 2009

038 - Yano, et al - Mesothelioma in a Worker Who Spun Chrysotile Asbestos at Home During Childhood - 2009

039 - Reserved

040 - Pesche, et al - Cancer Mortality in a Surveillance Cohort of German Males Formerly Exposed to Asbestos - Int. J. Hyg. Environ. Health - 2010

041 - Baumann, et al - Pleural Mesothelioma in New Caladonia, Associations with Environmental Risk Factors - 2011

042 - Skammeritz, et al - Asbestos Exposure and Survival in Malignant Mesothelioma, A Description of 122 Consecutive Cases at an Occupational Clinic - 2011

043 - Kanarek, M.S. - Mesothelioma From Chrysotile Asbestos - Update - 2011

044 -Testa, et al - Germline BAP1 Mutations Predispose to Malignant Mesothelioma - 2011

045 - Brief Amicus Curiae of Interested Physicians and Medical Researchers in Ford v. Boomer - 2012

Mesothelioma Index

Richard Cohen MD MPH
January 18, 2019

Mesothelioma Index

Richard Cohen MD MPH
January 18, 2019

061 - Loomis, et al - Cancer Mortality and Fibre Exposures Among North Carolina Asbestos Textile Workers - 2013

062 - Magnani, et al - Pleural Mesothelioma, Epidemiological and Public Health Issues. Report from the Second Italian Consensus Conference on Pleural Mesothelioma - 2013

062 - Magnani, et al - Pleural Mesothelioma, Epidemiological and Public Health Issues. Report from the Second Italian Consensus Conference on Pleural Mesothelioma - 2013

064 - Tarres, et al - Pleural Mesothelioma in Relation to Meteorological Conditions and Residential Distance from an Industrial Source of Asbestos - 2013

065 - Asbestos, Asbestosis, and Cancer, the Helsinki Criterial for Diagnosis and Attribution 2014, Recommendations - Scand J Wk Env Hlth - 2014

066 - Lacourt, et al - Occupational and Non-Occupational Attributable Risk of Asbestos Exposure for Malignant Pleural Mesothelioma - 2014

067 - Linton, et al - Factors Associated with Survival in a Large Series of Patients with Malignant Pleural Mesothelioma in New South Wales - 2014

068 - Offermans, et al - Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherlands Cohort Study - 2014

069 - Reid, et al - Mesothelioma Risk After 40 Years Since First Exposure to Asbestos - A pooled Analysis - 2014

070 - Simonsen, et al - Tuberculosis and Risk of Cancer, a Danish Nationwide Cohort Study - 2014

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Mesothelioma Index

Richard Cohen MD MPH
January 18, 2019

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Mesothelioma Index

Richard Cohen MD MPH
January 18, 2019

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Mesothelioma Index

Richard Cohen MD MPH
January 18, 2019

101 - Index of Articles Indicating that Smoking Does Not Cause Mesothelioma

BAP1 Tumor Genetics Sub-File

001 - Testa, et al - Germline BAP1 Mutations Predispose to Malignant Mesothelioma - 2011

002 - Carbone, et al - BAP1 Cancer Syndrome, Malignant Mesothelioma, Uveal and Cutaneous Melanoma, and MBAITs - 2012

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007 - Kadariya, et al - BAP1 Is a Bona Fide Tumor Suppressor, Genetic Evidence from Mouse Models Carrying Heterozygous Germline Bap1 Mutations - 2016

008 - Wang, et al - BAP1, Gene of the Month - 2016

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